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Letter to the Editor

In Reference to *Distinct Histopathology Characteristics in Empty Nose Syndrome*

Dear Editor:

We have read with great attention to the recent article "Distinct Histopathology Characteristics in Empty Nose Syndrome".[1]

In particular, we have appreciated the efforts of Ching-Lung et al. to define the histopathological and molecular patterns for the identification of this controversial syndrome. However, in our opinion, there are several critical issues to consider in relation to their study.

First, in the cohort of 17 enrolled patients with Empty Nose Syndrome (ENS), there is a significant percentage of subjects (35%) affected by allergic rhinitis that could be a confounding factor on the results. Previous studies have shown a decrease in nasal tactile sensitivity and have highlighted interesting aspects of the nasal chronic inflammatory condition in allergic rhinitis.[2] In these cases, in fact, the dysregulation of the innate and adaptive mucosal immunity related to allergy could influence the histological pictures and the expression of the mucosal receptors.

Second, the authors do not take into account, the nasal microbiota and the relationship between the presence of nasal dysbiosis and reactive nasal inflammation that could trigger a complex network of processes regulating mucosal permeability, TJ function, and the nasal neuroimmune system.[3]

Third, a recent retrospective review of 294 consecutive patients treated for sinonasal malignancies explained that turbinate resection is not correlated with nasal obstruction and poor sinonasal function. No cases of severe debilitating sinonasal dysfunction or ENS were reported.[4]

Fourth, it is not clear what is intended by the authors when they state that "the sensation of nasal patency closely related to thermoreceptor-like transient receptor potential channel melastatin 8 (TRPM8) in the nasal mucosa, which remained to play an implicit role in ENS": Along with mucosal histological change, the perception of nasal patency depends on adequate numbers and stimulations of the TRPM8 receptors. In fact, if this assumption were true, it is not clear how submucosal reconstruction over the lateral wall of the nasal cavity in these patients could resolve the nasal obstructive symptoms in the presence of an underexpression of these receptors. In summary, the authors' efforts to define and classify a syndrome that is still of uncertain pathogenesis and is not always repeatable after surgical demolition of the turbinates are certainly to be appreciated. However, in our opinion, the factors responsible for the so-called ENS are manifold and most likely related to the interaction of the nasal microenvironment with the mucosal firewall whose permeability causes the pathogenesis of this complex pathology.

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